Odontological Section.

February 26, 1912.

Mr. H. Lloyd Williams, President of the Section, in the Chair.

Two Cases of Hypoplasia of Enamel.

By J. G. Turner, F.R.C.S., L.D.S.

Two children, a boy and a girl, were exhibited, showing the early stage of enamel hypoplasia. In the boy the condition was a more or less general one—i.e., it extended symmetrically round a large number of the permanent teeth, and, so far as its position was concerned, it apparently referred to somewhere about the third year of life. As a matter of fact there was a history of severe measles in the third year, and, especially on the lower teeth, a distinct band of white-coloured enamel was to be seen marking the interference with the formation of the enamel; it was the first stage of enamel hypoplasia. The enamel was full-bodied, but wanting in molecular constitution. It could not be seen so well in the boy's upper teeth, but it was to be seen extending to the canines and perhaps the first premolars.

The second case, the girl, was exhibited for the purpose of showing the localized condition. The enamel hypoplasia was in the first stage, and the condition affected the left lower permanent central, lateral, and canine. The canine had a wonderful yellow colour on it which he could not explain; the patch on the front of the lateral was less yellow, and that on the front of the incisor dull white, but both lateral and canine also showed some dull white enamel. The enamel was also full-bodied. Obviously a local cause had to be sought. The history was very distinct. At the age of 3 the temporary teeth in that position were taken out for abscess, and, later, he had extracted all the remaining temporary teeth

for sepsis, so that the permanent teeth had been interfered with in their growth by the spread of inflammation from the septic temporary teeth. As far as the tooth itself was concerned the same result, the first stage of enamel hypoplasia, was brought about, but localized. It would be seen that on the coronal half of the canine the hypoplasia spread right round—i.e., the inflammation involved the whole of the growing enamel organ. Discrete patches were very often found on one surface of a tooth only, a surface often affected being the anterior surface of the incisors, because the roots of the temporary incisors were in near approximation to that surface. The inflammatory process must spread considerably to reach to the backs of the incisor teeth.

DISCUSSION.

The PRESIDENT (Mr. H. Lloyd Williams) said he had noted the dark and pigmented patches to which Mr. Turner had referred several times, and they were very difficult to understand. He did not know whether any of the Fellows had any experience of cutting sections from such teeth. He believed that Mr. Charters-White used to say that the white patches were as like as possible to composite rock—i.e., absolutely without a plan, as the enamel prisms had lost their shapes and were simply a jumble without any definite structure.

Mr. DOWSETT asked whether Mr. Turner looked upon the cause of the isolated patches in the temporary teeth in the girl as the same as the condition in the boy. The condition of the boy he understood was developmental, but in the girl he thought the isolated patches must be post-developmental, the result of a pathological change acting upon the enamel, because that portion of the enamel would be formed before the stage of inflammation in the temporary teeth. With regard to the discoloured patches, he had actually cut sections from two upper temporary incisors with brown patches upon them, and the microscopic appearance was very much the same as the ordinary appearance of enamel prisms, but the "brown striæ of Retzius" were very thick and well marked.

Mr. SIDNEY SPOKES said he had not seen the girl, but he understood from what Mr. Turner said that one cause of the trouble which he had described was abscess of a temporary tooth producing hypoplasia marks on the permanent successor. Some years ago, in a school of 800 children, he was very careful to take notes of all cases he came across where there was undoubted abscess of the temporary teeth and where the succeeding teeth, which he had the opportunity of seeing afterwards, were absolutely un-

blemished and free from any appearance of hypoplasia. That was particularly the case in bicuspids where the temporary molars were commonly abscessed, and the bicuspids afterwards were found to be quite free. The girl had, he thought, hypoplasia of the temporary teeth, which was distinctly more rare. Every authority that had dealt with hypoplasia was agreed upon the fact that temporary teeth were not so frequently affected as permanent teeth. With regard to the boy, he thought his lateral incisors had escaped, as they very frequently did in such cases. The general condition seen in hypoplastic front teeth was a mark on the central incisor, the tip of the canine, and the lateral incisors escaped. He was quite certain in his own mind that that was the usual experience in the ordinary well-developed cases of hypoplasia of the enamel. Mr. Turner now produced a case in which it was in its very early stage, so that some of his remarks perhaps would not hold good.

Mr. J. F. COLYER asked if Mr. Turner could explain why in that case there was a hypoplasia with the complete thickness of the enamel, while in other cases complete destruction occurred apparently of the enamel organ.

Mr. J. G. TURNER, in reply, said that he had not been able to make sections of the early stages of hypoplasia because he had never been heartless enough to extract any teeth of that kind. If anyone would send him such teeth he would be very pleased to do so. With regard to the cause of the isolated patches in the girl and the bands in the boy, they were practically identical—either circulating toxins or some interference with nutrition, the one absolutely local, the other absolutely general, affecting all the teeth, or portions of the teeth, being formed at that time. When once the enamel was formed there was no question of its being altered again. There were cases in which doubt might be raised with regard to whether the enamel had not been macerated in some way, but that only occurred when the teeth were retained in their dead state or were surrounded by purulent material. That, he believed, was not an explanation of the colour to which he referred, unless a *Staphylococcus aureus* infection were responsible for it. He rather thought that Mr. Dowsett and himself were not referring to the same point.

Mr. DOWSETT thought that the patch on the permanent teeth could not possibly be formed by an abscess on a temporary tooth because that portion of the enamel would be completely formed before the temporary tooth could have an abscess on it.

Mr. TURNER thought the question was rather the other way about, that the canine would not be completely formed till some considerable period after birth.

Mr. DOWSETT said he granted that was so with regard to the canine, but although he had not seen the child he had seen similar patches on the incisors.

Mr. TURNER, continuing, said there were patches on the two incisors and he was quite content that those were being formed before the age of 3. No

difficulty was experienced in understanding the matter if the way in which enamel is plastered on over a cusp in constantly increasing cones was borne in mind. There was also the fact that at the end of the process there might be additions of partial cones. That he had not been able to work out, but undoubtedly the whole outer layer approximately corresponded. In one of the incisor teeth of the boy two patches could be seen which were obviously formed at the same period. With regard to Mr. Sidney Spokes's objection that he examined a large number of cases and did not find the condition, he could only say that the histories were good in an astonishing number of cases if a practitioner only had sufficient material going through his hands. If he had not he could hardly expect to find them. It was possible to get not only the histories, but the actual material in situ. Personally he had models of the dead temporary tooth, the actual dead temporary tooth and the necrotic succeeding tooth in his possession. The range of injury varied from the specimen he had shown to absolute death and embraced a large number of quaint forms and monstrosities which had been described chiefly as dilaceration. There was a very good specimen showing the condition in the Museum of the Royal Dental Hospital of London in which the permanent teeth had been killed outright. With regard to the question of the complete thickness of the enamel, he presumed that depended entirely on the time at which the injury occurred and the extent of the injury. If the injury was extensive enough, then all that could be hoped for was that some enamel would be formed lower down where the growing enamel organ had just escaped. If the injury was more extensive the whole thing was killed. If the injury was not very extensive a little overlapping might be obtained, and sometimes a good enamel succeeding a bad enamel. If sections of hypoplastic teeth were taken after they were decalcified it would be found that a very thick Nasmyth's membrane appeared to be present; not a true Nasmyth's membrane, for under the microscope it showed the traces of enamel formation, in fact it showed very well that it was the organic matrix of enamel prism. It was the last despairing effort of the enamel organ to form enamel, but it had never got calcified, and remained as an acid-resisting membrana preformativa. There was every grade between absolute death and consequent want of enamel right up to fully formed but badly coloured enamel, and he thought soft enamel. White hypoplastic enamel was, he thought, softer than normal enamel.